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Diet, Tobacco, Alcohol, and Stress as Causes of Coronary Artery Heart Disease: An Ecological Trend Analysis of National Data

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The present investigation examined the temporal relationships between changes in coronary artery heart diesease (CAHD) mortality rates from whites (1938–1980) and changes in national measures of dietary elements, tobacco consumption, alcohol consumption, and unemployment. The magnitude and latency of the causal relationships were estimated with the use of cross-lagged correlation functions (CCFs) and Granger causality tests. Preliminary CCFs showed consistent correlational patterns between CAHD and tobacco, ethanol, and dietary fats. There was little association between CAHD and dietary cholesterol. Ethanol, tobacco, and the ratio of saturated to polyunsaturated fats (S:P) were analyzed for directional causality using Granger causality tests. The S:P ratio demonstrated a unidirectional Granger causal relationship with CAHD mortality in all sex and age groups. The estimated latency of this relationship was 23 to 30 years. This finding supports a causal relationship between diet, specifically fats, and the risk of CAHD two or three decades later.

The development of atherosclerosis is believed to be a slow, continuous process. Studies have shown that fatty deposits are present in the aortas of many children before the age of three and in the coronary arteries before the age of 20 [1,2]. Even though these deposits are known precursors of atherosclerotic lesions, a direct connection between these deposits in the young and their future development of fatal CAHD has not been proven in humans. Nevertheless, it is plausible that the development of coronary artery heart disease (CAHD) is gradual and that the rate of its development is determined by the amount of previous exposure to several of its putative causes.

Death from CAHD, then, may be attributable to a person's habits early in life. If so, then changes in CAHD mortality rates would show some relationship to changes in CAHD risk factors that occurred not simultaneously, but years, perhaps decades, earlier. Recent evidence suggests that certain habits and behaviors during young adulthood are associated with changes in CAHD risk factors [3]. Previous research has provided substantial evidence that diet [2,4,5,6] and cigarette smoking [7,8,9] contribute directly to the development of CAHD, and that other factors, such as stress [10,11] and alcohol [12,13,14], may also alter the risk of CAHD. Few risk factors have

Abbreviations: CCF: cross-lagged correlation function CAHD: coronary artery heart disease S:P ratio: ratio of saturated to polyunsaturated dietary fats

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been examined in the longitudinal context, however. The use of ecological series of data in the present study provided an opportunity to examine the relationships between CAHD and its possible causes as they exist outside laboratory or clinical settings. Logically, causes of CAHD should be detectable through the similarities across time between the trends of the causes and trends in CAHD mortality. Furthermore, the delay (lag) in a variable's effect on CAHD should be possible to estimate by calculating the number of years which separate similar changes in these trends.

Of the many major risk factors, three were examined indirectly as predictors of subsequent mortality from CAHD: smoking habits, stress, and components of diet (specifically as they relate to serum cholesterol). Longitudinal causal associations between these variables and CAHD mortality rates may offer a new perspective on reasons for the current decline in CAHD mortality rates.

MATERIALS AND METHODS

Sources of Data

Dependent Variables: Mortatlity rates are available through yearly volumes of *Vital Statistics of the United States* [15]. Each volume contains mortality data for all disease codes based on the International Classification of Diseases (ICD) for that year. From these records, several dependent variables were selected for use in the present analysis. A series of mortality data representing approximate CAHD death rates for whites was extracted using the disease codes in each year that were most comparable to the original "Diseases of the Coronary Arteries" (fourth revision of the ICD). Death rates for whites due to "Total Heart Disease" and "Hypertensive Heart Disease" were also extracted. One mortality code, "Accidental Death," believed to be unrelated to CAHD, was included to aid in the identification of spurious relationships.

All series of mortality rates were adjusted according to comparability ratios published after each revision of the ICD [16–20]. Numbers of deaths for each year were muliplied by comparability ratios from all subsequent revisions to set all values to a common revision of the ICD, usually the ninth revision. The adjustment scheme for all variables, which includes the specific disease codes and the comparability ratios applied in the present study, is shown in Table 1.

Revisions of the ICD disrupted the coding procedures for many subdivisions of overall "Diseases of the Heart." These abrupt changes in code have discouraged comparisons of data before and after specific revisions [21–24]. For CAHD-related codes, the most dramatic coding changes occurred in the sixth and eighth revisions. The category "Diseases of the Coronary Arteries" in the fourth (code 94b) and fifth (code 94a) revisions was most equivalent to "Arteriosclerotic Heart Disease specified as involving coronary arteries" (code 420.1) in the sixth and seventh revisions. In 1968, most cases from code 420.1 were transferred to the modern eighth and ninth revision code "Ischemic Heart Disease" (410). The adjustment process used in the present study removed most of the abrupt rate changes caused by periodic revisions in the coding of causes of death. In 1964 (in the middle of the seventh revision), however, many deaths were transferred abruptly into the CAHD code 420.1 from 420.0 ("Arteriosclerotic Heart Disease so described"). This large break was corrected by subtracting the number of deaths between 1963 and 1964 from the total number of deaths in 1964 and subsequent years.

TABLE 1
Adjustment Scheme for Mortality Rate Series[a]

Revision Number (dates)	Mortality Series	ICD Codes	Sex	Comparability Ratios 4–8	
				65–74 Years	45–54 Years
Fourth	THD	90–95	M	0.97	0.97
(1930–38)			F	0.97	0.97
Fifth	THD	90–95	M	1.07	1.05
(1939–48)			F	1.12	1.13
	CHD	94a	M	1.23	1.14
			F	1.37	1.29
	HHD	93d, 131a	M	0.26	0.29
			F	0.33	0.49
Sixth	THD	410–443	M	1.01	1.00
(1949–57)			F	1.00	1.00
	CHD	420.1	M	1.03	1.01
			F	1.04	1.01
	HHD	440–443	M	1.16	1.03
			F	1.04	1.01
Seventh	THD	400–402,	M	1.0045	1.0045
(1958–67)		410–443	F	1.0045	1.0045
	CHD[b]	420.1	M	0.89135	0.89135
			F	0.89135	0.89135
	HHD	440–443	M	0.398	0.398
			F	0.398	0.398
Eighth	THD	390–8, 402,	M	1.0126	1.0126
(1968–78)		404, 410–29	F	1.0126	1.0126
	CHD	410	M	1.0003	1.0003
			F	1.0003	1.0003
	HHD	402, 404	M	1.0	1.0
			F	1.0	1.0
Ninth	THD	See Eighth	M	1.0	1.0
(1979—)			F	1.0	1.0
	CHD	410	M	1.0	1.0
			F	1.0	1.0
	HHD	402	M, F	0.3028	0.3028
		404	M, F	0.8252	0.8252

[a]Mortality rates in each revision were multiplied by comparability ratios shown in that revision and all subsequent revisions. The total heart disease (THD) series and the coronary heart disease (CHD) series were adjusted to the ninth revision metric. Hypertensive heart disease (HHD) was adjusted to the eighth revision metric.

[b]After 1963, a constant was subtracted from all CHD rates (see text): males 65–72 (−365), males 45–54 (−49), females 65–74 (−169), and females 45–54 (−10).

Data from all available years were used for "Total Heart Disease" (1930–1980) and "Hypertensive Heart Disease" (1938–1980) series. CAHD mortality data from 1930 to 1939 were excluded, based on indications that the dramatic increase in CAHD mortality during this period was caused by increased recognition of the disease and not by an actual increase in the number of cases [25]. To examine mortality rates in their least aggregated form, mortality rates for specific age and gender groups were analyzed separately. Specifically, these groups included rates for 65–74-year-old males, 65–74-year-old females, 45–54-year-old males, and 45–54-year-old females.

Independent Variables: The term "independent variable" refers here to a variable's role as a possible cause. Some of the data on independent variables were collected from existing records. Many of these were yearly per capita consumption rates for specific foods or other products. The list included the consumption of fish, poultry, red meats, milk, cheese, eggs, margarine, lard, butter, total fats, tobacco, beer ethanol, wine ethanol, hard liquor ethanol, and total ethanol [26]. As it has been used in other studies [11,27], a series of unemployment rates was included as an indicator of stress. Unless otherwise indicated, all of the above series were obtained from volumes of *Statistical Abstracts of the United States* [28] and *Historical Statistics of the United States* [29]. All series were available for the period 1910 to 1980, except tobacco consumption (1920–1980), total fats (1931–1980), and ethanol measures (1934–1976).

The diet variables that are discussed in the present investigation were calculated using estimates based on existing data. This list included the estimated consumption of dietary cholesterol, polyunsaturated fats (more than one double bond between carbons; e.g., linoleic), saturated fats (fatty acids carrying the maximum number of hydrogens, all carbon chain lengths), and the ratio of saturated to polyunsaturated fats (S:P). References to this ratio in the literature often use the inverse expression (i.e., polyunsaturated: saturated); however, the S:P ratio was chosen to allow positive causal associations between this variable and mortality rates. Computational equations for these variables are shown in Table 2. As shown, the level of a particular dietary component was calculated by summing the estimated amounts of this component that were contributed from known per capita consumption of foods. For example, the level of polyunsaturated fat consumption in a given year was calculated by summing the total number of grams of polyunsaturated fats in the red meat, fish, poultry, eggs, milk, cheese, butter, lard, and margarine consumed per capita that year.

Transformations

Once developed, all independent and dependent series were transformed into units of yearly change, by first differencing (i.e., (year (t) − year ($t - 1$)), to remove long-term trends in the data. These first differenced series were used in all subsequent analyses.

Causal Analysis

Causal connections between mortality rates and other variables were examined through the analysis of lagged relationships among the differenced series. The lag in a relationship refers to the delay between a change in a causal variable and the resultant change in the caused variable. For example, a strong relationship between two variables at lag 10 (dependent variable lagged ten years *after* the independent variable) indicates a similarity between their trends after a delay of ten years.

Causal hypotheses were based on relationships between CAHD and other variables that have been established previously in the literature. These hypotheses were then tested, using cross-lagged correlations, followed by Granger causality tests (see below).

Several steps were taken to develop and support causal associations. First, the direction of causality was hypothesized and specified for all variables. In all cases, it was assumed that mortality rates were influenced by the independent variables chosen for this investigation. (The behavioral hypothesis that changes in mortality rates cause

TABLE 2
Estimation of Dietary Fats and Cholesterol from Various Sources in the American Diet[a]

Cholesterol (mg) = (Red meat [lb]) × (16 oz/lb) × (80.75 mg/3 oz) +
(Fish [lb]) × (16 oz/lb) × (55.38 mg/3 oz) +
(Poultry [lb]) × (16 oz/lb) × (70.25 mg/3 oz) +
(Eggs) × (240 mg/egg) +
(Milk [lb]) × (1.924 cups/lb) × (34 mg/cup) +
(Ice cream [lb]) × (16 oz/lb) × (56 mg/8 oz) +
(Cheese [lb]) × (16 oz/lb) × (24.33 mg/oz) +
(Butter [lb]) × (96 tsp/lb) × (12 mg/tsp) +
(Lard [lb]) × (96 tsp/lb) × (5 mg/tsp)

Saturated Fats (g) = (Red meat [lb]) × (16 oz/lb) × (5 g/3 oz) +
(Fish [lb]) × (16 oz/lb) × (2 g/3 oz) +
(Poultry [lb]) × (16 oz/lb) × (2 g/3 oz) +
(Eggs) × (2 g/egg) +
(Milk [lb]) × (1.924 cups/lb) × (5.1 g/cup) +
(Cheese [lb]) × (16 oz/lb) × (5 g/oz) +
(Butter [lb]) × (4 sticks/lb) × (57.3 g/stick) +
(Lard [lb]) × (32 tbs/lb) × (5 g/tbs) +
(Margarine [lb]) × (4 sticks/lb) × (17 g/stick)

Polyunsaturated Fats (g) = (Red meat [lb]) × (16 oz/lb) × (0.5 g/3 oz) +
(Fish [lb]) × (16 oz/lb) × (1.5 g/3 oz) +
(Poultry [lb]) × (16 oz/lb) × (1.5 g/3 oz) +
(Eggs) × (0.05 g/egg) +
(Milk [lb]) × (1.924 cups/lb) × (0.2 g/cup) +
(Cheese [lb]) × (16 oz/lb) × (.15 g/oz) +
(Butter [lb]) × (4 sticks/lb) × (2.1 g/stick) +
(Lard [lb]) × (32 tbs/lb) × (1 g/tbs) +
(Margarine [lb]) × (4 sticks/lb) × (25 g/stick)

Ratio S:P = Saturated fats (g)/Polyunsaturated fats (g)

[a]Quantities of cholesterol and fats within specific foods were obtained from Appendix D in *Nutrition:Concepts and Controversies* [33].

persons to change their habits was not considered in the present study.) As a rudimentary check for directional causality, inverse cross-lagged correlation functions (CCFs), where mortality preceded the independent variables (leads zero through 40 years), were examined.

A second causal argument was based on the expectation that the relationships between the independent variables and mortality rates (dependent variables) would be disease-specific. One independent variable should not correlate equally well with several disease mortality rates, and the observed differences should be plausible. To test this, the CCFs between all independent variables and mortality rates from accidents were examined. The consistency of findings across different age groups, within each sex, was also considered. Specifically, members of the same sex should suffer the effects of disease-causing agents after similar delays in time.

Additional support for causal inferences came from the specific patterns of CCFs generated by the analysis. If variable A contributed to the development of CAHD, the effect was likely to be noticeable after some delay in time, L; however, the duration of that delay probably varied around L as an average among individuals. Therefore, the pattern of cross-lagged correlation coefficients was expected to have a peak at lag L,

and gradually diminishing values as one moved away from lag L. Lag L in this case represented the latency period for this relationship that occurred most frequently in the population. The width of the non-zero subset of coefficients around lag L gave an indication of the range of likely latency periods. For example, if habit A contributes to the development of CAHD after a delay of approximately fifteen years (L), give or take five years, the CCF for this relationship would exhibit high positive cross-lagged correlation coefficients between lag 10 and lag 20 years.

The CCFs between each independent variable and the mortality rate series were examined for promising patterns. The variables that produced CCFs having no pattern, having several abrupt changes from strong positive to strong negative coefficients, or having inconsistent patterns across age groups were not included in subsequent procedures. Also, variables that exhibited strong correlations with accident mortality rates over several lags were disregarded.

Independent variables having both a plausible causal relationship with CAHD mortality and promising CCF patterns were further tested for unidirectional causality using the principles of Granger causality [30]. Basically, Granger's notion of causality states that variable X causes variable Y if one can predict present values of variable Y better by knowing previous values of X than by simply knowing previous values of Y [30,31]. The same argument follows for Y causing X. The test for Granger causality in the present study was performed by regressing the mortality series on to lagged series of mortality and lagged series of the independent variable under investigation. Next, the independent variable was regressed on to lagged values of itself and lagged values of mortality. Each regression procedure was performed twice, once with four lags and once with eight lags of the "predicted" variable in the prediction model. According to Granger's principles, one would expect that the addition of more previous information, by adding four additional lags to the equation, would only affect the strength of poor predictors. The particular lags of the "predictor" variable included in the models were chosen based on the correlation patterns in the associated CCFs.

RESULTS

Examination of the CCFs yielded the following general observations. None of the first differenced independent variables exhibited consistent correlational patterns with differenced accident rates. Cross-lagged correlation functions involving differenced total heart disease mortality rates frequently resembled the corresponding CCFs from CAHD mortality rates, although the latter functions were always more distinct (i.e., fewer sign changes, larger magnitude of correlation). Hypertensive heart disease mortality showed no consistent correlational pattern across sex and age groups except with beer ethanol and total ethanol.

Within the CAHD set, most correlational patterns were more distinct for male mortality series than for female mortality series and more distinct for older age groups than for younger age groups. As expected, not all independent variables exhibited a relationship with mortality rates. Unemployment rates, total fats, wine ethanol, and hard liquor ethanol demonstrated either no correlational pattern or severely inconsistent correlational patterns with all mortality series.

First differenced tobacco consumption correlated strongly positive with differenced CAHD mortality between lag 2 and lag 8 and strongly negative between lag 25 and lag 35. This pattern was relatively distinct for all males and older females. For all sex and age group mortality rates, both the ethanol from beer and total ethanol consumed

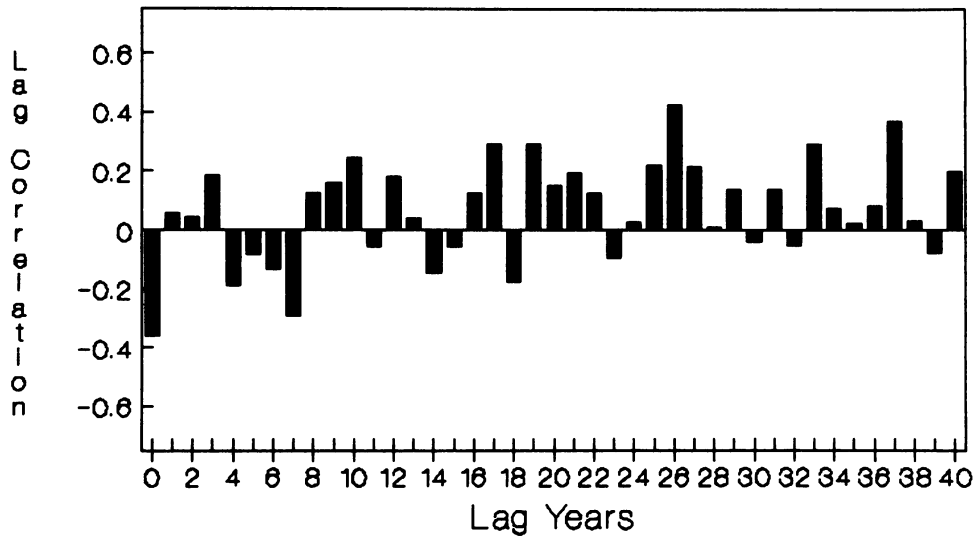

FIG. 1. Cross-lagged correlation coefficients between first-differenced CAHD mortality rates (females 45–54) and the ratio of saturated to polyunsaturated fats. Lag years indicate the delay between fat consumption and subsequent mortality rates.

produced several strong positive coefficients between lags 18 and 37. Surprisingly, the CCFs depicting the relationship between these ethanol measures and hypertensive heart disease exhibited coefficients that were of similar magnitude but of opposite sign to those involving CAHD, between lags 12 and 28.

Of the four variables computed to represent a dietary component of the entire set of foods, only one, first differenced dietary cholesterol content, showed little relationship to differenced CAHD mortality rates. Polyunsaturated fat content correlated consistently negative with CAHD mortality from lag 15 to lag 35 years (peaking at lags 19 and 26) for all age and sex groups. Saturated fat content produced primarily positive coefficients over the same lag range; however, the patterns of coefficients were much less distinct and of less magnitude than those associated with polyunsaturated fats.

The ratio of saturated fats to polyunsaturated fats (S:P) in the foods correlated positively with CAHD mortality over the range from lag 15 to lag 35 (see Figs. 1–4). Bimodal peak correlations were at lag 19 and 26 consistently. The relationship between this fat ratio and CAHD mortality exhibited the strongest and most distinct patterns with male mortality rates. Mortality rates for older females (Fig. 2) correlated in a pattern similar to the males, although slightly weaker. The pattern was barely discernible in the relationship between fat ratio and CAHD mortality for females 45–54 (Fig. 1). It should be emphasized that the observed correlations reflect relationships between yearly fluctuations (i.e., differences) in CAHD and yearly fluctuations in the variables under investigation. The correlations do not reflect coincidental long-term secular trends.

Four independent variables were tested for causality and directional causality using Granger's procedures: per capita consumption of tobacco, ethanol from beer, total ethanol, and the ratio of saturated to polyunsaturated fats. Of these variables, tobacco and both ethanol measures failed to demonstrate unidirectional causality. Neither ethanol measure provided stable predictors of CAHD mortality when eight lags of

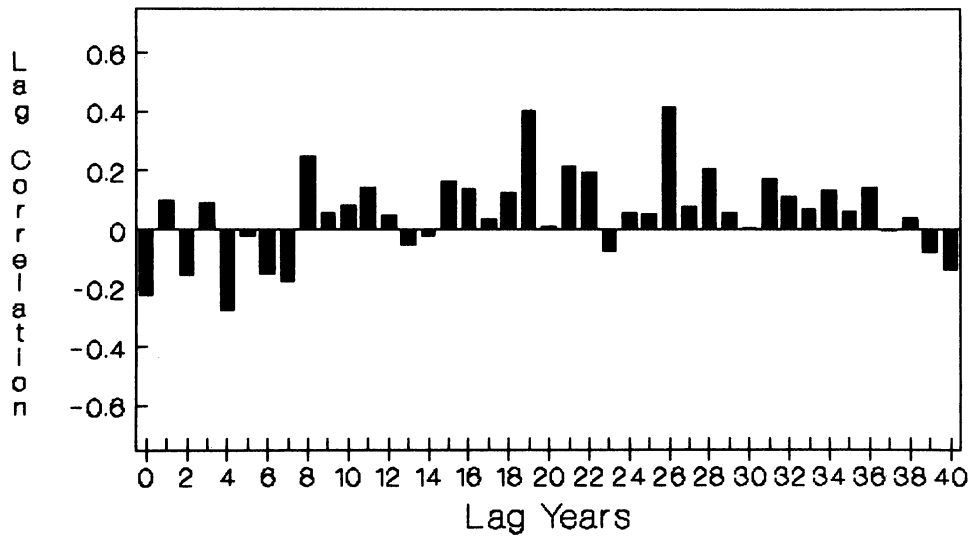

FIG. 2. Cross-lagged correlation coefficients between first-differenced CAHD mortality rates (females 65–74) and the ratio of saturated to polyunsaturated fats. Lag years indicate the delay between fat consumption and subsequent mortality rates.

mortality were included in the model. Lags 1, 5, and 9 of tobacco consumption were helpful predictors of CAHD mortality; however, unidirectional causality was refuted by the strength of lags 8 and 9 of CAHD mortality as predictors of tobacco consumption.

The ratio of saturated fats to polyunsaturated fats consumed by the average individual demonstrated a unidirectional "Granger causal" relationship with CAHD mortality rates in all age and sex groups (refer to Table 3). In all cases, at least one

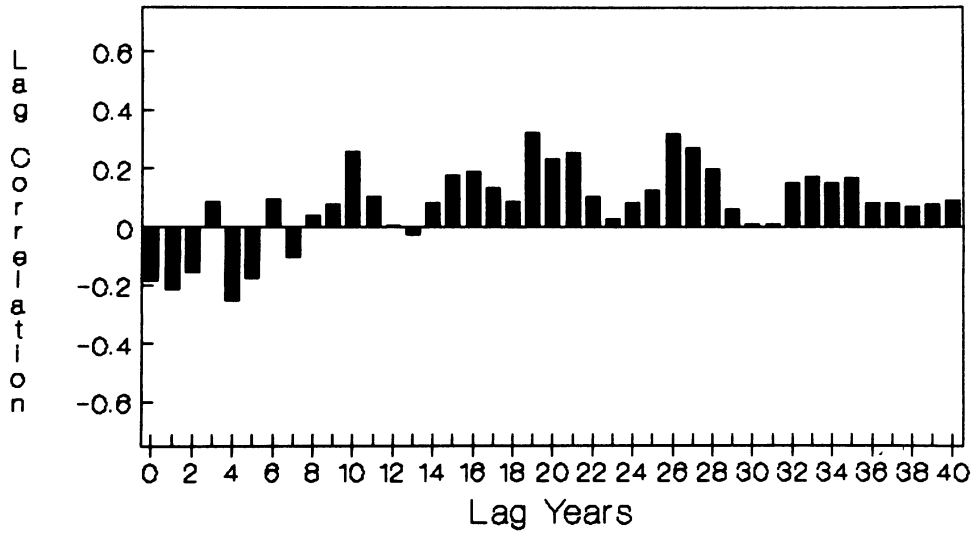

FIG. 3. Cross-lagged correlation coefficients between first-differenced CAHD mortality rates (males 45–54) and the ratio of saturated to polyunsaturated fats. Lag years indicate the delay between fat consumption and subsequent mortality rates.

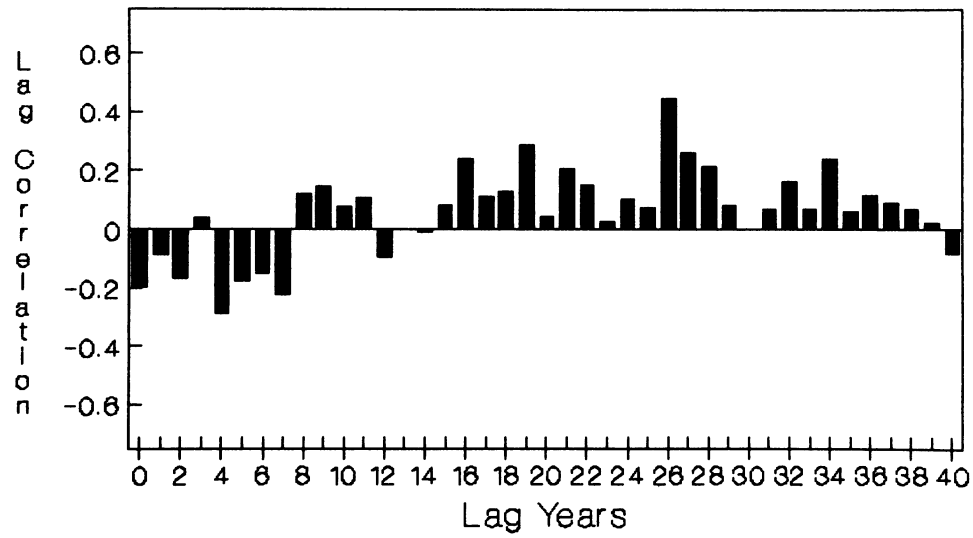

FIG. 4. Cross-lagged correlation coefficients between first-differenced CAHD mortality rates (males 65–74) and the ratio of saturated to polyunsaturated fats. Lag years indicate the delay between fat consumption and subsequent mortality rates.

TABLE 3
Results of Granger Causality Tests for the Ratio of Saturated to Polyunsaturated Fats and Coronary Heart Disease Mortality in All Sex and Age Groups

Predicted Variable	Lags of Predicted Variable in Equation	Predictor Variable	Lags of Predicted Variable in Equation	Predictor Lags with Significantly Non-Zero Beta Weights at $p < .10$	Overall F for Regression Model
Males 65–74					
CHD	1–4	Sat:Poly Fats	18–30	23, 24, 25, 26	**7.38
CHD	1–8	Sat:Poly Fats	18–30	24, 26, 27	**4.23
Sat:Poly Fats	1–4	CHD	1–12	9	1.98
Sat:Poly Fats	1–8	CHD	1–12	None	1.20
Males 45–54					
CHD	1–4	Sat:Poly Fats	18–30	26	**6.80
CHD	1–8	Sat:Poly Fats	18–30	25, 27, 28, 30	**15.60
Sat:Poly Fats	1–4	CHD	1–14	8, 9	*4.06
Sat:Poly Fats	1–8	CHD	1–10	None	1.85
Females 65–74					
CHD	1–4	Sat:Poly Fats	18–30	26, 28	**3.95
CHD	1–8	Sat:Poly Fats	18–30	26	2.07
Sat:Poly Fats	1–4	CHD	1–14	4	2.34
Sat:Poly Fats	1–8	CHD	1–10	None	0.88
Females 45–54					
CHD	1–4	Sat:Poly Fats	18–30	26, 29	**7.80
CHD	1–8	Sat:Poly Fats	18–30	26, 29	**4.28
Sat:Poly Fats	1–4	CHD	1–14	None	0.89
Sat:Poly Fats	1–8	CHD	1–10	None	0.91

*$p < .05$
**$p < .01$

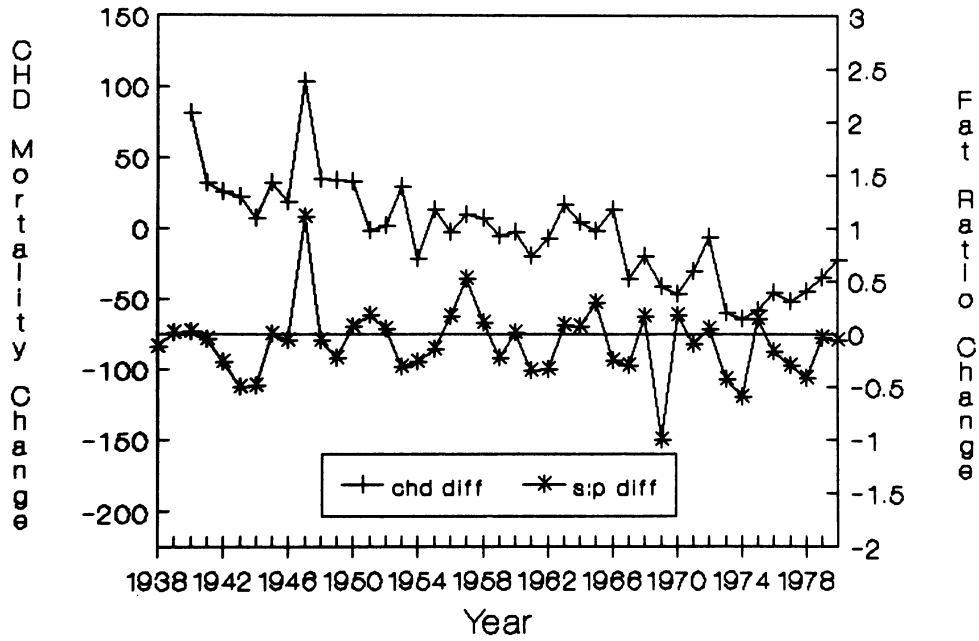

FIG. 5. An illustration of the similarities between the first-differenced series of CAHD mortality rates (males 65–74) for the years 1938 to 1980 and first-differenced series of the S:P ratio lagged by 26 years (1912–1954).

significantly non-zero beta weight was present for mortality rates lagged 23 to 30 years. In no instances was lagged mortality a significant predictor (i.e., "retrospective" predictor) of the fat saturation ratio. The strength and consistency of relationship between these variables indicates that the patterns of change in CAHD mortality should resemble the patterns of change in dietary fat consumption in previous decades. Similarities between the changes in the S:P ratio (the differenced series) and the corresponding changes in CAHD mortality (65–74-year-old males) 26 years later are shown in Fig. 5.

Beta weights for the variable included in the Granger causality equations predicting CAHD mortality rates (per 100,000) from the S:P ratio are shown in Table 4. Changes in CAHD mortality rates can be predicted reliably based on knowledge of the fat saturation ratio of the food consumed per capita 20 to 30 years earlier. For example, a nationwide reduction of two units (i.e., 7:1 to 5:1) in the ratio of saturated to polyunsaturated fats (from the food sources in this study) leads to the following predictions. Twenty-six years after this change, for each 100,000 persons in this age group, 146 fewer males 65–74, 64 fewer females 65–74, 8 fewer males 45–54, and 10 fewer females 45–54 would die from CAHD than had died in previous years. Twenty-seven years after this change in diet, 200 fewer individuals (per 100,000) from all four age groups would die from CAHD. The same computations can be applied to predict the latent effects of dietary fat saturation after a specific number of years.

LIMITATIONS

The nature and quality of the data in the present study warranted both a refutative analysis strategy and cautious interpretation of results. The analytical procedures

TABLE 4
Beta Weights for Lags of Coronary Heart Disease (CHD) and the Ratio of Saturated to Polyunsaturated Fats Predicting Coronary Heart Disease Mortality, from the Granger Causality Analysis

Variable	Lag	Males 65–74	Males 45–54	Females 65–74	Females 45–54
CHD	1	−0.29	0.39	−0.14	−0.19
CHD	2	−0.01	−0.06	0.22	−0.38
CHD	3	0.30	−0.22	0.56	−0.32
CHD	4	0.07	−0.15	0.19	0.34
CHD	5	0.09	0.35	−0.22	0.03
CHD	6	0.20	0.03	−0.04	−0.01
CHD	7	−0.03	0.07	−0.17	−0.07
CHD	8	−0.01	0.01	−0.19	−0.02
Sat:poly	18	10.09	1.94	0.39	−3.39[a]
Sat:poly	19	−23.22	2.28	5.47	−1.25
Sat:poly	20	−33.87	−5.76[a]	−0.14	−0.71
Sat:poly	21	−6.23	−0.32	−4.26	−2.90
Sat:poly	22	−6.22	−1.96	8.99	−1.70
Sat:poly	23	7.15	−6.05[a]	10.11	−0.28
Sat:poly	24	36.32[a]	2.50	13.48	−0.31
Sat:poly	25	31.60	6.58[a]	13.92	0.73
Sat:poly	26	73.74[a]	4.01	32.53[a]	5.42[a]
Sat:poly	27	72.82[a]	11.65[a]	13.89	3.80
Sat:poly	28	48.29	7.83[a]	7.96	3.13
Sat:poly	29	21.29	3.45	−8.52	4.20
Sat:poly	30	7.27	5.22[a]	−9.43	−0.57

[a]The beta parameter estimated by the regression procedure is significantly different from zero, $p < .10$.

presented in the Methods section (e.g., yearly differencing and Granger causality) were chosen to reduce the possibility that coincidental ecological trends would be mistaken for causal evidence. Despite these precautions, the reader should be aware of several limitations inherent in the data used for this investigation. First, the integrity of the mortality data was limited by the level of diagnostic sophistication in the medical community and the disease classification system used at that time. Comparability ratios could account for known technical changes in the coding of causes of death, but not for changes in the knowledge and skills of the medical practitioners or the extent to which new information had reached different areas of the country.

Second, the use of aggregated, national-level data required the assumption that one measure is somehow representative of the characteristics of an entire nation. Per capita mortality and consumption rates represent average rates for the population, but not necessarily typical rates. Furthermore, these rates could be particularly unrepresentative of the individuals under investigation. Specifically, those who developed CAHD may not have consumed diets similar to the diet represented by the average national rates.

Third, calculations of dietary fat variables were based on the limited number of known food sources for which consumption rates were available during the years from 1910 to 1980. These did not include many food sources that clearly contribute to the overall fat content in the American diet. Fat consumption information was limited to four categories, including total fat, butter, lard, and margarine. Without more specific

information about the rate of consumption of animal and vegetable fats, especially those used in processed foods, it is likely that rates of saturated and polyunsaturated fat consumption have been underestimated. The present findings rest on the assumption that the trend in the S:P ratio observed here represents the trend in the actual S:P ratio from all food sources.

Finally, in several cases the absence of definitive findings suggests that the available measures were not representative of their intended risk criterion variables. The prime example of this possibility is the use of tobacco consumption as an indicator of smoking behavior. Tobacco consumption is influenced both by changes in the number of smokers and the severity of their habits.

DISCUSSION

Although the S:P ratio in this study was based on a particular set of foods, the present findings indicate that the national trend toward eating foods that are higher in polyunsaturated fats, which began decades ago, contributed to the present decline in CAHD mortality for all age and sex groups. The estimated latency for the causal relationship between S:P and CAHD mortality is 23 to 30 years. The length of this temporal delay has strong implications for the importance of preventive health measures. The relationship shown in the present study suggests that eating habits early in life contribute to a lethal condition two or three decades later. The positive aspect of this finding is the extent to which this source of risk is alterable. Based on the regression data shown in Table 4, we can predict that each unit change in the S:P on average would result in about 12,500 fewer male deaths and 6,000 fewer female deaths during the five-year period 24–28 years later, in the 65–74-year age group alone.

A latency period of 23 to 30 years between dietary elements and mortality rates supports the hypothesis that CAHD develops graudally and implies that preventive alterations early in life, specifically in the types of dietary fats, could reduce significantly the number of deaths from CAHD in the decades to come. Evidently, adults in the 1970s and 1980s have benefited from dietary changes that occurred in their teen and early adult years.

The credibility of the relationship between CAHD mortality and the S:P ratio was supported by the findings of the Granger causality tests and by other considerations as well. First, specific consumption rates for foods high in saturated fats (e.g., butter and eggs) produced large positive cross-lagged correlation coefficients within the same latency period as S:P. Similarly, consumption rates for foods high in polyunsaturated fats (e.g., margarine) produced high negative cross-lagged correlation coefficients in the same lag period.

An additional endorsement was provided by the lack of association between the lagged consumption rate for dietary cholesterol and CAHD mortality. Dietary cholesterol has been shown to correlate highly ($r = .84$) with the amount of saturated fat in the diet [32], yet some researchers believe that it contributes little to serum cholesterol levels [33]. Thus, if the relationship between S:P and CAHD mortality were due to coincidental similarities in trend, the dietary cholesterol consumption rate (theoretically non-causal) could have been associated with CAHD mortality in a similar fashion. It was not.

Based on the present findings, there is reason to suspect a direct connection between the amount of saturated fats in a young American's diet and his chances of death from CAHD two or three decades later.

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